Annals of Clinical and Analytical Medicine
Original Research

Lipoprotein subfractions in patients with depression: The lipoprint system

Lipoprotein sub-fractions and depression

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Abstract
Aim: The aim of the study is to investigate the relationship between depression and changes in 25-OH Vitamin D and Lipoprotein serum levels, which have been suggested to be associated with Central Nervous System neurotransmission and certain psychiatric disorders.
Material and Methods: The study included a patient group consists of 40 depressive individuals who have applied to the Psychiatry outpatient clinic of our hospital and have been first diagnosed with depression according to SCID-1 (Structured Clinical Interview for DSM IV Axis I Disorders) criteria and had no psychiatric or systemic disease that could affect the result, as well as a control group of 40 healthy individuals with similar demographic characteristics. In the collected serum samples, 25-OH vitamin D, LDL, HDL, VLDL, total cholesterol, and TG levels were measured enzymatically and spectrophotometrically; Apolipoprotein AI and Bl00 levels were measured nephelometrically, LDL and HDL subfractions were measured using the Lipoprint System, and then the results were evaluated statistically.
Results: Atherogenic LDL 3 and LDL 4 were significantly higher in the patient group (p=0.001 and p=0.0155, respectively). The mean LDL particle diameter (LDL Mean) was significantly lower in the patient group (p=0.0017). There was no significant difference between the patient and control groups in terms of Buoyant LDL, but Small Dense LDL levels were significantly higher in the patient group (p=0.008 and p<0.001, respectively). LDL 1 Subfraction level of the patient group was statistically significantly higher than the control group (p = 0.0046). There was no significant difference in LDL 2 (p=0.3560).
Discussion: According to the results, LDL 3, LDL 4, and Small Dense LDL serum levels of the patients with depression were found to be higher. The fact that Small Dense LDL has a long circulation time in the blood, atherogenic and proinflammatory properties, better penetrability into the arterial intima layer and is considered as a risk factor in the CVD group, suggests that the risk of atherosclerosis, inflammation and CVD may be higher in patients with depression.

Keywords
Lipoprotein, 25-OH Vitamin D, Depression, Atherosclerosis, Small Dense LDL, LDL Subfractions, Buoyant LDL

DOI: 10.4328/ACAM.20815 Received: 2021-08-13 Accepted: 2021-10-19 Published Online: 2022-03-28 Printed: 2022-05-01 Ann Clin Anal Med 2022;13(5):470-474
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Introduction

The density of lipoprotein is related to the number of lipids and proteins in a particle [1]. The particle size decreases as the amount of cholesterol in the lipid core of particles decreases [2].

In ultracentrifugation, large and light lipoproteins accumulate on the top, and small and heavy ones at the bottom. As a result, chylomicrons (CMs) are classified into five groups: very low-density lipoproteins (VLDL), intermediate-density lipoproteins (IDL), low-density lipoproteins (LDL), and high-density lipoproteins (HDL) [3].

Recent studies have shown that HDL subgroup analysis is more determinant in the identification of coronary artery disease risk compared to total HDL measurement [4-6].

Another study identified three HDL subgroups such as large HDL (HDL-L), intermediate HDL (HDL-I) and small HDL (HDL-S) based on the analysis of normolipidemic and dyslipidemic plasma samples [7]. A significant difference was found between HDL-L and HDL-S subgroups in dyslipidemic and normolipidemic plasma samples. The development of atherosclerosis is inversely proportional to HDL-L and directly proportional to HDL-S [8].

LDL is divided into subgroups according to chemical composition, density and particle size. Small LDL particles are more atherogenic since they show reduced affinity for LDL receptors, greater binding to the endothelial proteoglycans, and better penetration to the arterial intima layer [9]. Today, several methods such as nuclear magnetic resonance, gel electrophoresis, lipoprint system, ultracentrifugation and high-performance gel filtration chromatography are used to determine LDL subgroups. However, the reference method in this regard is not certain [10].

Apoproteins are involved in the water solubility of lipoproteins as well as their recognition by cell receptors, thus facilitating their receptor-mediated endocytosis [3].

Vitamin D is synthesized in the skin from 7-dehydrocholesterol by UV irradiation at a wavelength of 290-310 nm in the form of cholecalciferol, which is the main source of endogenous Vitamin D [11].

Depression is not just a mental breakdown but a set of symptoms and signs named as a depression. It is a syndrome including symptoms such as slowness in thinking, speaking, and movements, stillness, unworthiness, guilt, tiredness, loss of attention and concentration, reluctance, loss of motivation, pessimist thoughts and feelings, and slowing of physical activities, in general, a deeply sad emotional state [12].

It was demonstrated that free fatty acids and cholesterol levels in the blood increase in emotional arousal in psychiatric disorders [15].

While it has been suggested that Vitamin D and depression are inversely proportional to each other, i.e. decreased Vitamin D levels increase the risk of depression; studies have shown that levels below 20 ng/ml cause depression [16].

In light of all this information, our study aims to investigate the relationship between depression and changes in 25-OH vitamin D and lipoprotein serum levels, which have been suggested to be associated with Central Nervous System neurotransmission and certain psychiatric disorders.

Material and Methods

Within the scope of this prospective study, a control group consisting of 40 patients (9 males (22.5%), 31 females (77.5%)) and 40 healthy volunteers (16 males (40%), 24 females (60%)) who applied to University Training and Research Hospital Psychiatric Clinic were included in the study. Ethical approval was obtained from Training and Research Hospital Local Ethics Committee and the study was carried out in accordance with the Helsinki Declaration.

The sample group included 40 patients who have applied to the Psychiatry outpatient clinic of our hospital with psychiatric complaints, and who have been first diagnosed with depression according to the SCID-I (Structured Clinical Interview for DSM IV Axis I Disorders) criteria (an interview tool that was structured by the interviewer and is applied to investigate the diagnosis of Axis I mental disorders and whose validity-reliability have been confirmed by Çorapoğlu et al. (1999)) [13], and who have not received any treatment yet and had no pathological disease other than depression, as well as 40 healthy controls. Venous blood samples were collected from the sample group of 80 individuals, between 8:00 am and 10:30 am, following a 12-hour fasting and a 15-minute rest, in vacuum tubes with gel and without anticoagulant. The tubes were left for at least 15 minutes to ensure that they were clotted and then centrifuged at 3000 rpm for 15 minutes. The serum samples were aliquoted and stored in a freezer at -20oC for the tests, which would not be run on the same day. Then, the samples were left at 2-8 degrees to thaw one day before the study day, and taken out from the refrigerator at an early hour and left to reach the study temperature.

25-OH Vitamin D levels were measured on the Architect i 2000 device using the 'Microparticle Enzyme Immunoassay Method' (Abbott Diagnostics, USA). Total cholesterol, LDL, VLDL, HDL, triglyceride levels were measured using the 'Enzymatic-Photometric Method' on the Architect ci6000 device (Abbott Diagnostics, USA). Apoprotein A, Apoprotein B, Lipoprotein (a) levels were measured on the Image 800 device using the "Nephelometric Method'' (Beckman Coulter, USA). Lipoprotein subfractions were run using the "Non-denaturing Polyacrylamide Gradient Gel Electrophoresis (PAGE)" lipoprint method. First, the samples were treated with "Sudan Black B", which binds the cholesterol in serum lipoproteins. At pH = 8.2-8.6, three mA current was applied on the tube heads during the electrophoresis that lasted nearly one hour. Lipoproteins were sorted by particle size in the form of bands stained with lipophilic stain.

Statistical analysis

'McdCalc' (version 12.3.0.0) statistical software was used to evaluate data from the sample group. The Kolmogorov-Simimov test was used to determine whether the data of the patient and control groups fit the normal distribution. The independent t-test was used to evaluate the data of the patient and control groups that were normally distributed. The Mann Whitney U test was applied to compare the patient and control groups not fitting the normal distribution. The significant difference between the groups was evaluated within the 95% confidence interval ($p < 0.05$). Box-Whisker plots were used for the graphical

representation of the patient and control group data.

Results

As a result of the statistical analysis of the analytes examined in our study, the p -values of the average, standard deviation, minimum, maximum and statistical significance level of the measurements and demographic information made in the healthy control group and patient group are shown in Table 1 and Table 2.

Table 1. Statistical Analysis of Demographic Characteristics of Patient and Control

	CONTROL (Mean ± SD)	PATIENT (Mean ± SD)	P
Age (Year)	31.9 ± 9.8	35.9 ± 13.8	0.139
Sex (M/F) (n)	16/24	9/31	0.074

Significant p < 0.05*

Table 2. Statistical Analysis of the Vit D and Lipoprotein Results in Patient and Control Groups

	CONTROL (Mean ± SD)	PATIENT (Mean ± SD)	P
25-OH Vitamin D(ng/ml)	14.080 ± 4.7898	18.690 ± 7.2383	0.0013
Total Cholesterol (mg/dl)	181.500 ± 30.107	176.650 ± 31.511	0.4836
Triglyceride (mg/dl)	108.400 ± 48.371	107.821 ± 47.165	0.9571
HDL (mg/dl)	47.825 ± 10.568	46.075 ± 8.564	0.4184
LDL (mg/dl)	111.975 ± 27.377	107.900 ± 28.347	0.5151
Apolipoprotein A-I (mg/dl)	114.222 ± 28.815	121.697 ± 28.671	0.2423
Apolipoprotein B100 (mg/dl)	60.993 ±19.214	66.387 ± 19.951	0.2218
HDL Small (%)	6.775 ± 2.106	7.425 ± 2.205	0.1816
HDL Intermediate (%)	24.600 ± 4.348	24.300 ± 4.158	0.7534
HDL Large (%)	16.050 ± 7.313	14.625 ± 5.172	0.3178
LDL 1 (%)	39.275 ± 10.145	32.550 ± 10.478	0.0046
LDL 2 (%)	19.500^1	21.600 ± 8.949	0.3560
LDL 3(%)	1.950 ± 3.411	4.450 ± 3.928	0.0001
LDL 4(%)	0.075 ± 0.349	0.525 ± 1.198	0.0155
LDL MEAN (A°)	271.193 ± 3.002	268.903 ± 3.633	0.0017
Buoyant LDL* (%)	58.275 ± 16.318	52.025 ± 15.624	0.0810
Small Dense LDL* (%)	1,925 ± 3.590	7.125 ± 11.780	<0.001

H Calculated using the formula developed by Vega et al. [23]. Significant p < 0.05*

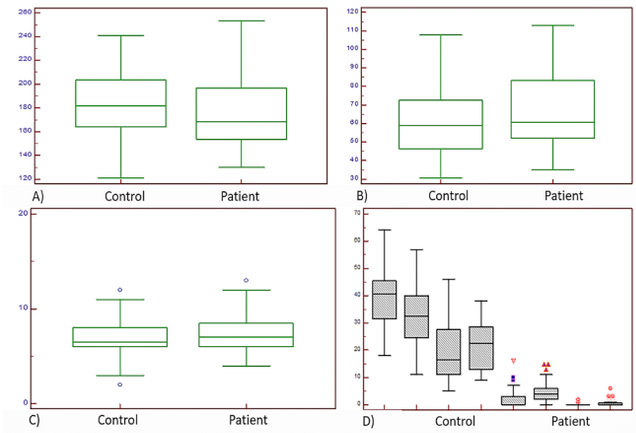

Figure 1. Analysis of Lipoprotein Results in Patient and Control Groups A) Statistical analysis graph of lipid parameters results, B) Statistical analysis graph of Apolipoprotein B-100 results, C) Statistical analysis graph of HDL sub-fractions results, D) Statistical analysis graph of LDL dub-fractions results

When both groups were statistically evaluated in terms of socio-demographic characteristics, no significant difference was found (P>0.05).

The calculation is made as follows, if the mean LDL diameter is below 163°A:

LDL 1 Buoyant LDL

LDL 2 + LDL 3 + LDL 4 + LDL 5 + LDL 6 + LDL 7 = Small Dense LDL

And, as shown below, if the mean LDL diameter is above 163°A:

LDL 1 + LDL 2= Buoyant LDL

LDL3 + LDL 4 + LDL 5 + LDL 6 + LDL 7=Small Dense LDL.

In the results obtained from the patient group and the control group, even 25-OH Vitamin D levels were low (below 20ng/ml) in both groups, the results of the patient group were not considered statistically significant (p=0.001). No statistically significant difference was found between total cholesterol, TG, HDL, LDL, Apolipoprotein B-100, and HDL subfraction results (p>0.05). Examining LDL subfractions results, they were not significantly higher in the control group (p=0.0046). There was no significant difference in LDL 2 (p=0.35). Atherogenic LDL 3 and LDL 4 were significantly higher in the patient group (p=0.001, p=0.0155, respectively). The mean LDL particle diameter (LDL Mean) was significantly lower in the patient group (p=0.0017). There was no significant difference between the patient and control groups in terms of Buoyant LDL (p=0.008), but Small Dense LDL values were significantly higher in the patient group (p<0.001).

Discussion

In this study, we aimed to investigate the relationship between depression and the changes in 25-OH Vitamin D and Lipoprotein serum levels, which have been to be suggested to be associated with Central Nervous System neurotransmission and certain psychiatric disorders.

While it is accepted that there is an inverse relationship between Vitamin D, whose receptors in the Central Nervous System have been shown and efficiency have been discussed, and depression risk, i.e. decreased serum Vitamin D levels increase depression risk, the study conducted by Milaneschi et al. [21] showed that levels below 20 ng/ml cause depression [22]. As a result of our enzymatic and spectrophotometric measurements, serum Vitamin D levels were below 20 ng/ml in both groups, but were statistically significantly higher in the patient group than in the control group, which was contrary to the literature (p=0.0013). While this is considered significant for the patient group, this lower value in the control group may be associated with the low number of samples in the patient and control group included in the study, independent of the symptoms of depression.

In the studies conducted by Melin et al. [15], and Chang et al. [16], it was demonstrated that free fatty acids and cholesterol levels in the blood increase with emotional arousal in psychiatric disorder. Maes et al. [20] found that in depressive patients and their first-degree relatives, blood cholesterol levels as well as esterified cholesterol rates were lower than in the normal group, and suggested that less esterification of cholesterol may lead to an increase in the cell membrane fluidity, thus leading

to depression.

The relationships between total cholesterol in serum and depressive disorders have been investigated, but their relationship with other lipids and depressive disorders has been rarely discussed. The ratio of serum HDL level to total cholesterol was reported to be statistically significantly lower in patients with major depression compared to controls [15]. There are studies showing that serum LDL levels are significantly higher in women in the group consisting of patients with major depressive disorder compared to the control group, while it is significantly lower in men in the same group [21]. In the meta-analysis of 36 studies and conducted by Jane et al. [22], serum LDL was generally significantly lower. While in some studies TG levels were found to be higher in patients with major depressive disorder [23]; no significant difference was found in the study by Özcankaya et al [18].

In our study on this controversial issue, we investigated total cholesterol, TG, VLDL, HDL and LDL levels in the serum samples collected from the patient and control groups. We did not find a significant difference between the two groups as a result of the statistical comparison of the depressive patient and healthy control groups ($p>0.05$). The oxidation of Apo B-100 increases atherosclerosis with the formation of several pro-inflammatory products [24]. Due to these characteristics, Apolipoprotein A-I and Apolipoprotein B-100 were chosen among many types of apolipoproteins, and their serum levels were measured nephelometrically. However, no statistically significant difference was found in serum Apolipoprotein A-I and Apolipoprotein B-100 levels between the patient and control groups ($p>0.05$).

Among the methods used for the determination of lipoprotein subfractions, the Lipoprint System is the first and only system approved by the FDA for the quantitative and qualitative measurement of LDL and HDL subgroups. Now, it is possible to measure small dense LDL, IDL, and HDL subgroups with high atherogenicity, which cannot be measured with other methods. In lipoprint, which is a linear polyacrylamide gel electrophoresis, lipoprotein particles are resolved with the effect of electric current without using the gradient gel of the gel matrix. Lipoprotein distribution is consistent with the distribution performance of the 'Continuous Gradient Ultracentrifugation' method [25].

Large HDL is inversely proportional to atherosclerosis, while small HDL has a positive relationship with atherosclerosis, since it infiltrates within the vessel wall [24]. In our study, HDL subfractions were classified in 10 subgroups. These 10 subgroups were divided into three sub-classes; the first three were Large HDL, the next four were intermediate HDL, and the last three were small HDL. As a result of the measurements with the lipoprint method, no significant difference was found in terms of HDL subfractions between the depressive patient group and the healthy control group in all of the three subgroups ($p>0.05$). The fact that there was no significant difference in Apolipoprotein A-I level supported our finding in the current study ($p>0.05$).

In the study by Vega et al. [18], LDL subfractions were divided into two classes, such as small dense LDL and Buoyant LDL; in the calculation, the controversial LDL 2 was associated with LDL diameter; the LDL 2s of the LDLs with a mean diameter above 263°A were named as Buoyant LDL. LDLs 2s with a mean diameter above 263°A were included in the small dense LDL class. This is caused by the fact that Small Dense LDL is extremely prone to lipid peroxidation, although it is less involved in the oxidative stress pathway of Buoyant LDL [25]. In our study, LDL subfractions were divided into seven subgroups. Among these subgroups, LDL 1 was found to be significantly higher in the control group; there was no significant difference in terms of LDL 2 in both groups, but LDL 3 and LDL 4, whose atherogenicity was shown to be high, were found to be significantly higher in the patient group. Given the diameters of LDL subfractions, the mean diameter in the patient group was found to be significantly smaller than the control group. Atherogenic small dense LDL subfractions were found to be significantly higher in the patient group compared to the control group, which may increase the risk of susceptibility to lipid peroxidation and exposure to atherogenicity in these lipoprotein fractions in the depression group.

Conclusion

The finding of such a difference in LDL subfractions, although there was no significant difference between the control and patient groups for all other lipoprotein and apolipoprotein levels, is explained by the fact that Small Dense LDL particles show increased affinity for LDL receptors, greater binding to the endothelial proteoglycans, susceptibility to lipid peroxidation, and better penetration to the arterial intima layer. Small Dense LDL has a long circulation time in the blood, atherogenic and proinflammatory properties, better penetrability into the arterial intima layer, and is considered a risk factor in CVD group, which suggests that the risk of atherosclerosis, inflammation and CVD may be higher in patients with depression.

Thus, higher Small Dense LDL levels in the patient group suggest that the risk of atherosclerosis may be higher in depressed individuals.

Limitations of the study

Although the study has been carefully prepared, we are still aware of its limitations and shortcomings. Firstly, as discussed above, our study population carries limited possibilities for exploring causal relationships and does not provide clinical follow-up, therefore it may not adequately reflect the general population and the relationship between depression and lipoprotein. Secondly, the number of patients and control groups can be increased, and gender differences can be examined, resulting in a wide range of data for Turkish society for males and females.

Acknowledgment

In memory of our esteemed teacher Prof. Dr. Hasan EFE, who contributed to this article and passed away during the publication phase.

Scientific Responsibility Statement

The authors declare that they are responsible for the article's scientific content including study design, data collection, analysis and interpretation, writing, some of the main line, or all of the preparation and scientific review of the contents and approval of the final version of the article.

Animal and human rights statement

All procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committee and with

the 1964 Helsinki declaration and its later amendments or comparable ethical standards. No animal or human studies were carried out by the authors for this article.

Funding: *None*

Conflict of interest

None of the authors received any type of financial support that could be considered potential conflict of interest regarding the manuscript or its submission.

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How to cite this article:

Tarkan Yazıcı, Süheyl Uçucu. Lipoprotein subfractions in patients with depression: The lipoprint system. Ann Clin Anal Med 2022;13(5):470-474